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#20	Search spla2 inhibitor and vascular	16:12:54	<u>18</u>
#19	Search spla2 inhibitor and amyloid	16:12:19	<u>1</u>
#2	Search spla2 inhibitor and hypertension	16:12:12	<u>4</u>
#18	Search spla2 inhibitor and vasoactive	16:12:05	<u>0</u>
#1	Search spla2 inhibitor	16:11:56	<u>205</u>
#10	Search beta amyloid and hypertension	16:08:45	<u>90</u>
#4	Search spla2 and hypertension	15:57:37	<u>4</u>
#9	Search amyloid and hypertension	15:02:13	<u>401</u>
#7	Search secretory phospholipase A2 and alzheimers	13:55:45	<u>2</u>
#6	Search secretory phospholipase A2	13:55:37	<u>968</u>
#5	Search secretory phospholipase A2 and hypertension	13:55:25	<u>4</u>
#3	Search spla2 inhibitor and vasospasm	13:54:41	<u>0</u>

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EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	65	"5700816"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 11:21
L2	56	"spla2 inhibitor"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 11:22
L3	8	"spla2 inhibitor" and "alzheimer"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 11:24
L4	0	secretory adj phospholipase adj inhibitor	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 11:47
L5	0	"secretory phospholipase inhibitor"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 11:47
L6	8	secretory near phospholipase near inhibitor	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 11:48
L7	65	"5654326"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 12:27
L8	1	"spla2 inhibitor" and "hypertension"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 12:50

EAST Search History

L9	0	"spla2 inhibitor" and "vascular disease"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 13:53
L10	0	"spla2 inhibitor" and "vasospasm"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 13:53
L11	10	"6,166,017"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 14:11
L12	4	"6642236"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 14:11
L13	14	"cortisol inhibitor"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 14:12
L14	3	"6642236" and "cortisol inhibitor"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 14:12
L15	0	oleyloxyethylphosphocholine	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT	OR	OFF	2008/01/25 16:43

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NEWS 20 DEC 04 LINPADOCDB now available on STN
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E1 1 OLEYLONITRILE/CN
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E11 1 OLEYLSTEARYLAMINE/CN
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E16 1 OLEYLTRIMETHYLENEDIAMINE/CN
E17 1 OLEYLTRIOCTADECYLAMMONIUM BROMIDE/CN
E18 1 OLF-1/EBF TRANSCRIPTION FACTOR (CAENORHABDITIS ELEGANS STRAIN N2
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E19 1 OLF-1/EBF-LIKE-1(8) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
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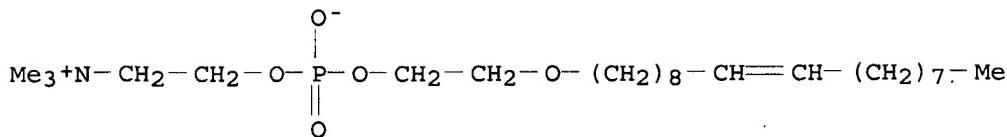
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E21 1 OLF-1/EBF-LIKE-2 (OS) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
SHORT ISOFORM 03) /CN
E22 1 OLF-1/EBF-LIKE-3 TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1) /CN
E23 1 OLFACTOMEDIN (HUMAN CLONE DE10316701-SEQID-623 GENE OLFM1
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E24 1 OLFACTOMEDIN (RANA CATESBEIANA PRECURSOR REDUCED) /CN
E25 1 OLFACTOMEDIN 1 (HUMAN GENE OLFM1) /CN

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E3 0 --> OOEPC/CN
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OOG4) /CN
E5 1 Oogenesin (MOUSE CLONE C0103) /CN
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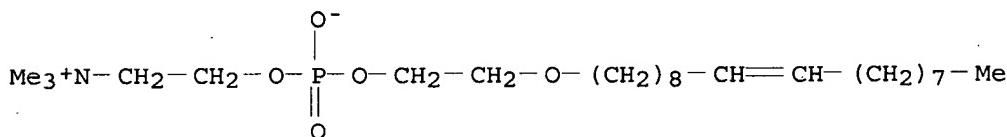
L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2008 ACS on STN
RN 96720-06-8 REGISTRY
CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-,
inner salt, 4-oxide (9CI) (CA INDEX NAME)
MF C25 H52 N O5 P
LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MEDLINE,
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DT.CA CAplus document type: Journal
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L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2008 ACS on STN
 RN 96720-06-8 REGISTRY
 ED Entered STN: 09 Jun 1985
 CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-, inner salt, 4-oxide (9CI) (CA INDEX NAME)
 MF C25 H52 N O5 P
 LC STN Files: BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MEDLINE, TOXCENTER
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=> d 13 1-8 ibib, abs, hitstr

L3 ANSWER 1 OF 8 MEDLINE on STN

ACCESSION NUMBER: 2002725402 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12489129
TITLE: Eicosanoids in insect immunity: bacterial infection stimulates hemocytic phospholipase A2 activity in tobacco hornworms.
AUTHOR: Tunaz Hasan; Park Youngjin; Buyukguzel Kemal; Bedick Jon C; Nor Aliza A R; Stanley David W
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska, Lincoln 68583-0816, USA.
SOURCE: Archives of insect biochemistry and physiology, (2003 Jan) Vol. 52, No. 1, pp. 1-6.
JOURNAL code: 8501752. ISSN: 0739-4462.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 19 Dec 2002
Last Updated on STN: 4 Mar 2003
Entered Medline: 3 Mar 2003

AB Intracellular phospholipase A(2) (PLA(2)) is responsible for releasing arachidonic acid from cellular phospholipids, and is thought to be the first step in eicosanoid biosynthesis. Intracellular PLA(2)s have been characterized in fat body and hemocytes from tobacco hornworms, *Manduca sexta*. Here we show that bacterial challenge stimulated increased PLA(2) activity in isolated hemocyte preparations, relative to control hemocyte preparations that were challenged with water. The increased activity was detected as early as 15 s post-challenge and lasted for at least 1 h. The increased activity depended on a minimum bacterial challenge dose, and was inhibited in reactions conducted in the presence of oleyoxyethylphosphorylcholine, a site-specific PLA(2) inhibitor. In independent experiments with serum prepared from whole hemolymph, we found no PLA(2) activity was secreted into serum during the first 24 h following bacterial infection. We infer that a hemocytic intracellular PLA(2) activity is increased immediately an infection is detected. The significance of this enzyme lies in its role in launching the biosynthesis of eicosanoids, which mediate cellular immune reactions to bacterial infection.

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L3 ANSWER 2 OF 8 MEDLINE on STN
ACCESSION NUMBER: 2002147899 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11841807
TITLE: Identification of the phospholipase A(2) isoforms that contribute to arachidonic acid release in hypoxic endothelial cells: limits of phospholipase A(2) inhibitors.
AUTHOR: Michiels Carine; Renard Patricia; Bouaziz Najat; Heck Nathalie; Eliaers Francois; Ninane Noelle; Quarck Rozenn; Holvoet Paul; Raes Martine
CORPORATE SOURCE: Laboratoire de Biochimie et Biologie Cellulaire, Facultes Universitaires Notre Dame de la Paix, 61 rue de Bruxelles, 5000, Namur, Belgium.. carine.michiels@fundp.ac.be
SOURCE: Biochemical pharmacology, (2002 Jan 15) Vol. 63, No. 2, pp. 321-32.
JOURNAL code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 8 Mar 2002
Last Updated on STN: 3 Apr 2002
Entered Medline: 27 Mar 2002

AB Changes in endothelium functions during ischemia are thought to be of importance in numerous pathological conditions, with, for instance, an increase in the release of inflammatory mediators like prostaglandins. Here, we showed that hypoxia increases phospholipase A(2) (PLA(2)) activity in human umbilical vein endothelial cells. Both basal PLA(2) activity and PG synthesis are sensitive to BEL and AACOCF3, respectively, inhibitors of calcium-independent PLA(2) (iPLA(2)) and cytosolic PLA(2) (cPLA(2)), while OPC, an inhibitor of soluble PLA(2) (sPLA(2)) only inhibited the hypoxia-induced AA release and PGF(2alpha) synthesis. Hypoxia does not alter expression of iPLA(2), sPLA(2) and cPLA(2) and cycloheximide did not inhibit PLA(2) activation, indicating that hypoxia-induced increase in PLA(2) activity is due to activation rather than induction. However, mRNA levels for sPLA(2) displayed a 2-fold increase after 2 hr incubation under hypoxia. BAPTA, an intracellular calcium chelator, partially inhibited the AA release in normoxia and in hypoxia. Direct assays of specific PLA(2) activity showed an increase in sPLA(2) activity but not in cPLA(2) activity after 2hr hypoxia. Taken together, these results indicate that the hypoxia-induced increase in PLA(2) activity is mostly due to the activation of sPLA(2).

L3 ANSWER 3 OF 8 MEDLINE on STN
ACCESSION NUMBER: 2001301904 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11226404
TITLE: The involvement of phospholipase A(2) in ethanol-induced gastric muscle contraction.
AUTHOR: Sim S S; Choi J C; Min D S; Rhie D J; Yoon S H; Hahn S J; Kim C J; Kim M S; Jo Y H
CORPORATE SOURCE: Department of Pathophysiology, College of Pharmacy, Chung-Ang University, 221 Huksuk-dong, Dongjak-gu, Seoul 156-756, South Korea.
SOURCE: European journal of pharmacology, (2001 Feb 16) Vol. 413, No. 2-3, pp. 281-5.
Journal code: 1254354. ISSN: 0014-2999.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 4 Jun 2001
Last Updated on STN: 4 Jun 2001
Entered Medline: 31 May 2001

AB To understand the underlying mechanism of ethanol in tonic contraction, the effect of ethanol on phospholipase A(2) and phospholipase C activities and the effects of phospholipase inhibitors on ethanol-induced contraction of cat gastric smooth muscle were tested. Circular muscle strips (2.0 x 0.2 cm) obtained from the fundus of cat stomach were used to measure isometric contraction. Ethanol elicited tonic contraction and activated phospholipase A(2) activity in a dose-dependent manner. Phospholipase A(2) inhibitors, manoolide (0.1--10 microM) and oleyloxyethyl phosphorylcholine (1--10 microM), significantly inhibited ethanol-induced contraction. Furthermore, 342 mM ethanol-induced contraction was significantly inhibited by cyclooxygenase inhibitors, ibuprofen (10--100 microM) and indomethacin (10--100 microM), but not by lipoxygenase inhibitors. On the other hand, phospholipase C inhibitors had no effect on ethanol-induced contraction, indicating that phospholipase C is not involved in ethanol-induced contraction. It is suggested from the above results that ethanol-induced contraction in cat gastric smooth muscle is, in part, mediated by phospholipase A(2) and cyclooxygenase pathways.

L3 ANSWER 4 OF 8 MEDLINE on STN
ACCESSION NUMBER: 2000106581 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10643787
TITLE: Novel strategies for opposing murine microglial activation.
AUTHOR: Paris D; Town T; Mullan M

CORPORATE SOURCE: The Roskamp Institute, University of South Florida, Tampa
33613, USA.. dparis@com1.med.usf.edu
SOURCE: Neuroscience letters, (2000 Jan 7) Vol. 278, No. 1-2, pp.
5-8.
Journal code: 7600130. ISSN: 0304-3940.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 9 Mar 2000
Last Updated on STN: 9 Mar 2000
Entered Medline: 22 Feb 2000

AB Pathologic microglial activation is believed to contribute to progressive neuronal damage in neurodegenerative diseases by the release of potentially neurotoxic agents, such as pro-inflammatory cytokines including tumor necrosis factor alpha (TNF-alpha). Using cultured N9 microglial cells, we have examined the regulation of TNF-alpha following endotoxic insult with lipopolysaccharide (LPS), focusing on the role of the pro-inflammatory phospholipase A2/mitogen activated protein kinase/arachidonic acid/cyclo-oxygenase-2 cascade and the nitric oxide/cGMP pathway. Data show that various inhibitors of the PLA2 cascade markedly inhibit LPS-induced TNF-alpha release, supporting a key role of this pathway in the regulation of microglial activation. We also investigated the putative effects of cGMP-elevating agents on blocking microglial activation induced by LPS. Data show that each member of this class of cGMP-elevating compounds that we employed opposed microglial TNF-alpha release, suggesting that strengthening intracellular cGMP signaling mitigates against microglial activation. Taken together, our results suggest novel strategies for reducing microglial activation.

L3 ANSWER 5 OF 8 MEDLINE on STN
ACCESSION NUMBER: 1999318341 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10391457
TITLE: Involvement of axonal phospholipase A2 activity in the outgrowth of adult mouse sensory axons in vitro.
AUTHOR: Hornfelt M; Ekstrom P A; Edstrom A
CORPORATE SOURCE: Department of Animal Physiology, Lund University, Sweden.
SOURCE: Neuroscience, (1999) Vol. 91, No. 4, pp. 1539-47.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 10 Sep 1999
Last Updated on STN: 10 Sep 1999
Entered Medline: 24 Aug 1999

AB The effect on axonal outgrowth of inhibition of phospholipase A2 activity was studied in a recently developed in vitro model, where dorsal root ganglia with attached spinal roots and nerve stumps from young adult mice were cultured in an extracellular matrix material (Matrigel). The phospholipase A2 inhibitors 4-bromophenacyl bromide and oleyloxyethyl phosphorylcholine dose-dependently reduced axonal outgrowth from the sciatic nerve stump. A similar inhibitory effect was seen when only the cut nerve end was exposed to the inhibitors in a compartmental culture system. The local effect of phospholipase A2 inhibition was further investigated on axons established in culture, using time-lapse recording. Exposure to phospholipase A2 inhibitors caused the retraction of filopodia extensions and a reduction in growth cone motility within a few minutes. After removal of inhibition, normal growth cone motility and axonal growth were regained. Nerve cell bodies and axons, in contrast to Schwann cells, showed immunoreactivity after staining with an antiserum against secretory phospholipase A2, and elevated levels of the enzyme could be detected

after culture for 24 h. The immunoreactive protein was of approximately 170,000 molecular weight (phospholipase A2-170) as determined by sodium dodecyl sulphate-polyacrylamide gel electrophoresis and immunoblotting. The localization of phospholipase A2-170 in axons growing into the Matrigel was also demonstrated by use of a whole-mount technique. The results of this study show the importance of continuous phospholipase A2 activity for growth cone motility and axonal outgrowth in the mammalian peripheral nerve, and suggest the involvement of an axonally localized enzyme.

L3 ANSWER 6 OF 8 MEDLINE on STN
ACCESSION NUMBER: 1999217849 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10203186
TITLE: Role of endothelial factors in the specific response of mouse tumour-feeding arterioles to stimulation of 5-HT₁ receptors.
AUTHOR: Laemmeli E; Stucker O; Vicaut E
CORPORATE SOURCE: Dept de Biophysique et INSERM U141, Hopital F. Widal, Paris, France.
SOURCE: International journal of radiation biology, (1999 Mar) Vol. 75, No. 3, pp. 365-71.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 11 May 1999
Last Updated on STN: 11 May 1999
Entered Medline: 29 Apr 1999

AB PURPOSE: To investigate the possible role of endothelial mediators on the increased vasoconstriction to 5-HT₁ receptor stimulation by the host-modified arterioles feeding a Meth-A tumour implanted in the flank of female Balb/c mice. MATERIALS AND METHODS: Using intravital microscopy, the response to the topical administration of the general 5-HT₁ agonist 5-carboxamidotryptamine maleate (5-CT; 10(-6) M to 10(-4) M) by the tumour-feeding arterioles with the responses of tumour-independent arterioles and those of control arterioles from mice without tumour after antagonization or inhibition of the synthesis of endothelial mediators was compared. RESULTS: The dramatically higher vasoconstriction to 5-CT observed in tumour-feeding arterioles than in tumour-independent or control arterioles still persisted when either nitric oxide synthase, cyclooxygenase, lipoxygenase, or phospholipase A2 were inhibited or when thromboxane A2 or endothelin were antagonized. CONCLUSIONS: It was concluded that the higher reactivity to 5-HT₁ stimulation by tumour-feeding arterioles is not due to changes in endothelial mediator release but probably due to changes affecting arteriolar smooth muscle.

L3 ANSWER 7 OF 8 MEDLINE on STN
ACCESSION NUMBER: 91183640 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1901255
TITLE: Inhibitors of cytochrome P-450 attenuate the myogenic response of dog renal arcuate arteries.
AUTHOR: Kauser K; Clark J E; Masters B S; Ortiz de Montellano P R; Ma Y H; Harder D R; Roman R J
CORPORATE SOURCE: Department of Physiology, Medical College of Wisconsin, Milwaukee 53226.
CONTRACT NUMBER: HL-29587 (NHLBI)
HL-33833 (NHLBI)
HL-36279 (NHLBI)
+
SOURCE: Circulation research, (1991 Apr) Vol. 68, No. 4, pp. 1154-63.
Journal code: 0047103. ISSN: 0009-7330.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 26 May 1991
Last Updated on STN: 3 Feb 1997
Entered Medline: 8 May 1991

AB The role of cytochrome P-450 in the myogenic response of isolated, perfused renal arcuate arteries of dogs to elevations in transmural pressure was examined. The phospholipase A2 inhibitor oleyloxyethylphosphorylcholine (1 and 10 microM) inhibited the greater than threefold increase in active wall tension in these arteries after an elevation in perfusion pressure from 80 to 160 mm Hg. Inhibition of cyclooxygenase activity with indomethacin (1 or 10 microM) had no effect on this response. The cytochrome P-450 inhibitors ketoconazole (10 and 100 microM) and beta-diethyl-aminoethylidiphenylpropylacetate (SKF 525A, 10 and 100 microM) also inhibited the myogenic response. At a pressure of 160 mm Hg, SKF 525A (10 microM) and ketoconazole (100 microM) reduced active wall tension in renal arteries by approximately 70%. Partial inhibition of the myogenic response was obtained after perfusion of the vessels with mechanism-based inhibitors of P-450, 1-aminobenzotriazole (75 microM) and 12-hydroxy-16-heptadecenoic acid (20 microM). The thromboxane receptor antagonist SQ 29,548 (1 or 10 microM) had no effect on the pressure-induced increase in active wall tension in renal arteries. Arachidonic acid (50 microM) constricted isolated perfused renal arteries and potentiated the myogenic response in the presence of indomethacin. This response was completely reversed by ketoconazole (100 microM) or SKF 525A (100 microM). Microsomes (1 mg/ml) prepared from small renal arteries (200-500 microns) and incubated with [1-14C]arachidonic acid (0.5 mu Ci, 50 microM) produced a metabolite that coeluted with 20-hydroxyeicosatetraenoic acid (20-HETE) during reversed-phase high-performance liquid chromatography. The formation of this product was inhibited by both ketoconazole and SKF 525A at concentrations of 10 and 100 microM. These results are consistent with the involvement of the vasoconstrictor 20-HETE and other cytochrome P-450 metabolites of endogenous fatty acids in the myogenic response.

L3 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

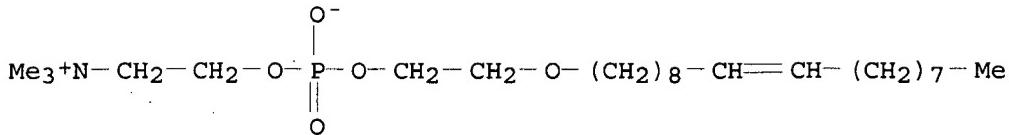
ACCESSION NUMBER: 1985:406131 CAPLUS
DOCUMENT NUMBER: 103:6131
ORIGINAL REFERENCE NO.: 103:1103a,1106a
TITLE: A new efficient and versatile synthesis of alkyl phosphorylcholines
AUTHOR(S): Magolda, R. L.; Johnson, P. R.
CORPORATE SOURCE: Cent. Res. Dev. Dep., E. I. du Pont de Nemours and Co., Wilmington, DE, 19898, USA
SOURCE: Tetrahedron Letters (1985), 26(9), 1167-70
CODEN: TELEAY; ISSN: 0040-4039
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 103:6131

AB Phosphorylcholines ROP(O)(O-)OCH₂CH₂N+Me₃ [R = Me(CH₂)_n, Me(CH₂)₇CH:CH(CH₂)₈, Me(CH₂)_mS(CH₂)₃, Me(CH₂)₇CH:CH(CH₂)₈S(CH₂)₃, Me(CH₂)_mOCH₂CH₂, Me(CH₂)₇CH:CH(CH₂)₈OCH₂CH₂; m = 15,17; n = 5,7,11,17] were prepared in 35-50% overall yield by treating ROH with POCl₃, followed by ethylene glycol and treating the resulting cyclic phosphates with Me₃N.

IT 96720-06-8P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)

RN 96720-06-8 CAPLUS

CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-, inner salt, 4-oxide (9CI) (CA INDEX NAME)



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FULL ESTIMATED COST	15.53	22.96
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=> E "OOPC"/CN 25
E1      1      OOLONGTHEANIN-3'-O-GALLATE/CN
E2      1      OOMYCIN A/CN
E3      0 --> OOPC/CN
E4      1      OOPG 1000/CN
E5      1      OOPG 1002/CN
E6      1      OOPLASM SPECIFIC PROTEIN (MUS MUSCULUS STRAIN NIH/SWISS GENE
OP1)/CN
E7      1      OOPODIN/CN
E8      1      OOPODIN, 11,13-DIDEHYDRO-/CN
E9      1      OOPORPHYRIN/CN
E10     1      OORA SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE OORA)/CN
E11     1      OORA SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(HELICOBACTER HEPATICUS STRAIN ATCC51449 GENE OORA)/CN
E12     1      OORAKKU APO 101/CN
E13     1      OORAKKU APO 101-HITALOID 3083-70B-MDI COPOLYMER/CN
E14     1      OORAKKU APO 101-HITALOID 3083-70B-MILLIONATE MR 200 COPOLYMER/CN
E15     1      OORAKKU APO 301/CN
E16     1      OORB SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE OORB)/CN
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E17 1 OORB SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(HELCOBACTER HEPATICUS STRAIN ATCC51449 GENE OORB)/CN
E18 1 OORC SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE OORC)/CN
E19 1 OORC SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(HELCOBACTER HEPATICUS STRAIN ATCC51449 GENE OORC)/CN
E20 1 OORD SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE OORD)/CN
E21 1 OORD SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(HELCOBACTER HEPATICUS STRAIN ATCC51449 GENE OORD)/CN
E22 1 OORP (ONCORHYNCHUS MYKISS OOCYTE)/CN
E23 1 OOSPGLYCOL/CN
E24 1 OOSPOALDEHYDE/CN
E25 1 OOSPOALDEHYDE, (2,4-DINITROPHENYL) HYDRAZONE/CN

=> d his

(FILE 'HOME' ENTERED AT 16:45:32 ON 25 JAN 2008)

FILE 'REGISTRY' ENTERED AT 16:45:44 ON 25 JAN 2008
E "OLEYLOXYETHYLPHOSPHOCHOLINE"/CN 25
E "OOEPC"/CN 25

L1 1 S 96720-06-8/RN
L2 1 S L1

FILE 'MEDLINE, CAPLUS, WPIDS, USPATFULL' ENTERED AT 16:49:58 ON 25 JAN
2008

L3 8 S L1

FILE 'REGISTRY' ENTERED AT 16:52:52 ON 25 JAN 2008
E "OOPC"/CN 25

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